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VETERINARY JOURNAL

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Obituary

THE LATE MR. A. A. TINDALL.

By the death of Mr. A. A. Tindall, at the ripe age of 91, the veterinary profession has lost not only a staunch supporter, but a good and loyal friend, as it is not too much to say that the firm of Baillière, Tindall & Cox, of which Mr. Tindall was the senior partner, has for many years been its mainstay for professional literature, both in regard to text-books and journalistic effort.

There are still some of the profession who will recollect his regular attendances at the opening ceremonies of the London College, and, on more than one occasion, as a guest at the R.C.V.S. dinners. Fleming, Smith, Robertson, Nunn, and many others now passed away, numbered him amongst their friends, and his acquaintance with the literature of the profession extended over sixty years.

In human medicine, Mr. Tindall published *The Medical Press and Circular*; and, in addition to his multifarious duties, he still found time to take up the responsibilities of London life, being a Freeman of the City, and a Member of the Drapers' Company.

To the end Mr. Tindall played an active part in the business, and to him must be credited his share in putting at the disposal of the profession, not only this journal (which the is oldest British veterinary periodical), but also the many standard text-books and monographs which were issued during his lifetime.

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THE VALUE OF RESEARCH IN COMPARATIVE MEDICINE.

THE article which we publish in full in this month's issue, by the courtesy of the Editor of *The British Medical Journal*, on "The Causation of Stone in India," by Colonel Robert McCarrison, is of mutual interest to the members of the two branches of medicine, the human and the veterinary; as the subject of the formation of calculi in the urinary tract is of equal importance to both—not only from the curative point of view, but also from the preventive.

Colonel McCarrison had, in India, the opportunity to study this disease from several different angles, especially in relation to the question of dietary; and the veterinary clinician, who numbers amongst his patients those who are vegetable feeders only, as well as the carnivores and those who are omnivorous, will read the details of the dietetic experiments with fascinating interest. On page 415, Dr. McCarrison tells us that one of his main objects in his feeding observations on rats was to endeavour to ascertain the faults in Indian dietaries to which "stone" might be attributable. This question equally applies to other countries than India, and to animals as well as men. Between pages 415 and 430 will be found paragraph after paragraph which will repay careful study by veterinary clinicians, especially those whose practice includes dogs and cats whose lives are luxurious and sedentary—for urinary calculus is so frequently observed in lap dogs and castrated cats. No paper has ever demonstrated more clearly the necessity for actual experimental research, combined with the value of collaboration between the workers in disease as it appears from the comparative aspect in both animals and man.

A SPECIAL VETERINARY PHYSIOLOGY NUMBER.

THE next issue of THE VETERINARY JOURNAL will be devoted largely to Physiological questions of veterinary interest. It is a reproach to our profession that we have no veterinary graduate who can lay claim to being an authority on this all-important subject, and that the Chairs of Physiology in our veterinary colleges are occupied by other than members of our own profession. It is certainly not for want of subjects which need to be studied, as they are legion, and the field is crying for workers. Continental colleges have for years been ahead of us in this matter, and the name of Colin is a household word wherever comparative physiology is mentioned; and as regards Great Britain, it is time that we too set our own house in order, and prepared ourselves for the opportunities which will be certain to be there when our colleges are admitted within the universities.

General Articles

"PULPY KIDNEY DISEASE" OF YOUNG LAMBS IN NORTH WALES.

By R. F. MONTGOMERIE, B.Sc., Ph.D., F.R.C.V.S.,
AND
W. T. ROWLANDS, M.R.C.V.S., D.V.S.M.
School of Agriculture, University College of North Wales, Bangor.

DURING each spring since 1924, the senior writer has observed considerable loss among young forward lambs within his province. Locally these deaths are commonly ascribed to "strike"—a term which really signifies sudden death—or to the effects of a wool-ball in the fourth stomach.

Investigations as conducted during the past five years resolve themselves into two parts: Preliminary work prior to season 1931 was directed towards determining whether a single disease condition was responsible for all, or the major part of, this loss. It was early evident that a clinical entity, which may be termed "pulpy kidney disease," was regularly present. This condition, as it appears in North Wales, is briefly described in the first part of this article. In the second part a preliminary attempt to determine its nature is detailed.

PART I.

Description of "Pulpy Kidney Disease" in North Wales.

Occurrence.—Loss from this disease is almost entirely confined to lambs three to six weeks old. The affected lambs are regularly the most forward of the flock. In North Wales the incidence of the disease is greatest among lambs which reach the susceptible age during the latter days of March and throughout April. In each year there is a short period of two or three weeks, varying slightly from year to year, during which almost all loss occurs.

Lambs of various breeds and crosses appear equally susceptible, and although the majority of the deaths occur among lowland flocks producing early fat lambs, the condition is not unknown on upland pastures. Some districts and certain farms are recognised as particularly liable to these losses. Many of these flocks suffer a loss of even from 4 per cent. to 6 per cent. through "strike," but the incidence

of the disease varies markedly from year to year, and the approximate average loss among lowland farms is 1 per cent. per annum. Not uncommonly deaths occur in batches, and a flock may suffer its total annual loss in the course of a single week.

Very commonly a sudden change to weather conditions conducive to good growth of grass is immediately followed by a crop of cases. In a cold " slow " spring, such as that of 1931, the incidence of the disease is reduced. Occasionally one or two cases occur after a light morning frost, but it does seem that a sudden change to quick growth is the predominant factor. The feeding of concentrates to ewes with lambs at foot is carried out only to a limited extent in North Wales, and lamb deaths are common in flocks not hand fed.

Symptoms.—Sudden death at any time of the day or night is most characteristic of the disease. In these cases some signs of a slight struggle and of the passage of a small quantity of diarrhœic fæces may be evident. Not uncommonly an affected lamb is seen alive; but, although death may be delayed four hours or longer, the duration of the illness is generally very short. The earlier symptoms appear to be inco-ordination and spasmodic contractions of the limbs. The lamb falls to the ground and lies quite flat on one side, legs extended, head retracted, respirations laboured, pupils dilated, salivating and convulsed at intervals by general tremors. Death takes place in the course of one of these spasms. A very occasional recovery has been reported to the writers, and in these cases early purgation has been achieved.

Lesions.—General congestion of the abdominal viscera is regular. The small intestine, particularly the jejunal portion, is often considerably congested. Although patchy congestion of the mucous and submucous coats is not uncommon, ulceration has not been observed. The contents in more severe cases may be somewhat blood-tinged. The mesenteric lymphatic glands are regularly enlarged and exude much fluid on section. The abomasum commonly contains one or more balls of wool and in the majority of cases the lambs have evidently commenced to nibble grass.

The most characteristic and obvious lesion is that of the kidneys, which are soft in consistency and have a curious blotched appearance. The renal cortex is always markedly congested and frequently broken down to a pulpy mass within the capsule. This pulpy state is rarely marked in examination made soon after death. Great congestion of the cortex, occasionally accompanied by hæmorrhages, is certainly present before actual death. The two kidneys may not be involved to an equal extent.

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The pericardial sac commonly contains an excess of fluid. Often this excess is considerable, and in examinations which have been delayed it may contain a large clot. Tiny sub-epicardial hæmorrhages are often evident, particularly along the course of the cardiac vessels, and much sub-endocardial hæmorrhage is regular—usually a large patch in the right ventricle. Congestion of the lungs, varying much in severity, is a usual feature. There is no lesion of the musculature.

Bacteriological Observations.—Records relating to twenty cases examined within one hour of death were accumulated prior to 1931. In all these cases aerobic and anaerobic media were inoculated with peritoneal fluid, liver, pericardial fluid and heart blood, and occasionally with kidney, mesenteric gland and spleen. With the exception of occasional growth from peritoneal fluid, these media regularly remained sterile, and it appeared evident that the condition was not due to bacterial invasion.

Field Observations on Prevention.—There is a firm belief in this province that blood letting does tend to prevent these losses. It has, therefore, become the practice in some farms to dock all the lambs at an early age, and this does seem to reduce the losses.

" Strike " in lambs was a serious problem in certain areas of this province now affected with lamb dysentery. Since 1928, it has been the practice of farmers owning infected flocks in these districts to inoculate all lambs with lamb dysentery serum within twelve hours of birth. During each of these seasons it has been remarkable that only a very occasional death from " strike " has been encountered on these farms. Several flocks have not lost a single lamb during the past three seasons, whereas previously there were regular losses, and whereas neighbouring flocks, not affected with lamb dysentery and not using serum, have had their usual losses.

Discussion.—This work appears to establish the disease responsible for the great majority of the losses among young forward lambs in North Wales as identical with the pulpy kidney disease described and investigated in New Zealand by Gill.[1] It differs from the pulpy kidney disease seen in England by Stewart [6] and by McKenna,[5] in that it is confined to much younger lambs and lambs not receiving concentrates. The writers are aware, however, that deaths similar to those met in North Wales are common in many parts of Great Britain. The condition described here is, however, markedly different from the " grass-ill " of young forward lambs prevalent in the North of England, and, according to the investigations of Stewart and Henderson,[7] caused by an organism indistinguishable from *V. septique.*

PART II.

The Nature of " Pulpy Kidney Disease " of Young Lambs.

In North Wales these deaths are commonly ascribed to the wool ball or wool balls so regularly present in the abomasum of dead lambs. In post-mortem examinations by the writers there was nothing in the appearance of the abomasum or in the postion of the wool balls which would suggest that they were of etiological significance. Moreover, wool balls are of very common occurence in young lambs in North Wales. W. Norman Jones, B.Sc., working in this College in 1925, made a record of the incidence of wool balls in early lambs killed for human consumption. One or more wool balls were present in 75 per cent. of these lambs.

Farmers commonly say " the lamb was doing too well." They ascribe any beneficial effect early docking may have to the check which this operation gives the lamb. While the condition of the lamb does appear to be a major predisposing factor, it is intimately associated with another influence. The forward state of the lamb and a sudden flush of young grass appear intimately bound in setting of the stage for pulpy kidney disease. It is accepted that rapidly growing young herbage is a particularly rich food and that its protein content is considerable. The change to a very nutritious diet may have its influence through an increase in the amount or an alteration in the composition of the milk of the ewe. It may, perhaps, act in some cases on ingestion by the lamb.

Stewart[6] investigated a theory that high protein diet was responsible for the pulpy kidney disease of lambs twelve to fifteen weeks of age occurring in the eastern counties of England. He concluded that high protein diets are not in any way responsible for this condition.

Since Gill commenced his investigation of pulpy kidney disease in New Zealand in 1926 no details of all his work have been published, but in popular accounts he has stated tentative conclusions at intervals [1 2 3]. His work has shown that the disease is not due to the invasion of the animal body by an infective agent. He has attained, much evidence to support his contention that dietary conditions are a predisposing factor. For some time he has held the view that the disease was a toxæmia of intestinal origin and in 1929 he[3] expressed his contention thus :—

" The most probable train of events, culminating in the death of the lambs, seems to be that the condition is due to toxins which are formed in, and absorbed from, the gut ; that these toxins may be produced in the gut by bacteria which are common and constant

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inhabitants of the gut, and which normally lead a quiet harmless existence there ; that through some factor or factors connected with the diet these bacteria have been enabled to increase in numbers and to produce larger quantities, or possibly more powerful types, of toxins. This by no means precludes the possibility that such organisms may have simultaneously increased in virulence so that they now attack types of lambs that were previously immune."

One of the writers (R. F. M.) had the advantage of personal discussion of this problem with Gill while the latter was visiting this country during the summer of 1929. Toxæmia of intestinal origin had suggested itself in the North Wales work. The post-mortem picture in pulpy kidney disease was very suggestive of either a septicæmia or a toxæmia, and, in face of the negative bacteriological findings, the latter is indicated. The lesions of the intestinal wall, together with the absence of evidence of a local infection, make a theory intestinal toxæmia feasible. The field observation that the use of serum in the prevention of lamb dysentery appears to entirely or almost entirely eliminate loss from " strike " added interest to this view. It seemed unlikely that this was a specific protection, since strike losses might be expected even five weeks after the inoculation.

It was decided to investigate the theory of toxæmia of intestinal origin during the 1931 season in North Wales, and although the disease was then of particularly low incidence, some interesting observations have been made.

The Toxicity of Intestinal Contents from Affected Lambs.— It was the writers' intention to restrict their investigation to the intestinal contents of cases seen alive or obtained within one hour of death, but, when it became apparent that the incidence of the disease in 1931 would be low and few such cases available, material from lambs dead a longer period was included in this work. In view of this use of less fresh material, opportunity was taken to test for toxicity the intestinal contents of a lamb twenty-four hours after death from, apparently, some other condition ; but it has not yet been possible to work with further lambs dead from other diseases or killed for investigation.

In each case the contents of the small intestine were collected in a sterile jar as soon as possible after death. They were thoroughly shaken with twice their volume of sterile physiological saline and thereafter centrifuged at 2,000 r.p.m. for one and a-half hours. The supernatant was filtered through a Seitz filter, fitted with an E. K. (special) pad. The filtrate regularly proved sterile. The diluted filtered intestinal contents thus obtained (hereafter termed " intestinal

filtrate ") were tested for toxicity by intravenous injection into rabbits.

Lamb R 1.—March 24th, 1931. Five weeks old lamb dead one hour after first seen affected. Post-mortem examination made one and a-half hours after death. Diagnosed as pulpy kidney disease. Intestinal filtrate injected in 1 c.c. dose to a 2,000 gm. rabbit caused death in convulsions five minutes later, and was similarly toxic to a 3,000 gm. rabbit in fifteen minutes.

Lamb L 1.—April 2nd, 1931. Four weeks old lamb found affected one hour previously ; examined immediately after death ; showing typical post-mortem lesions. One c.c. intestinal filtrate injected into a 2,000 gm. rabbit caused convulsions and death in eight minutes.

Lambs F 1, *PG* 1, *C* 1, *C* 2.—April 8th, 9th, 11th and 13th, 1931. These cases, 3, 5, 5 and 3 weeks old respectively, were typical cases of pulpy kidney disease and were all examined within two hours of death. In each case intestinal filtrate was injected in a 1 c.c. dose without obvious effect. Unfortunately, larger doses were not tested, but this experience determined the writers to employ a larger test dose in future work.

Lamb G 1.—April 15th, 1931. Five weeks old lamb found dead and examined about six hours after death. There was little evidence of putrefaction and characteristic lesions. The injection of 5 c.c. of intestinal filtrate caused the death in convulsions of a 2,500 gm. rabbit in two minutes. A dose of 1 c.c. proved fatal to two similar rabbits in four minutes.

Lamb M 1.—April 18th, 1931. Three weeks old lamb dead approximately twelve hours when examined. Despite post-mortem changes, and since typical lesions were seen, the toxicity of the intestinal contents was tested. The injection of 5 c.c. having produced no effect in twenty minutes, 8 c.c. were injected intravenously. Severe convulsions were quickly followed by death fifteen minutes after the second dose.

Lambs C 3, *C* 4.—April 18th and 21st, 1931.—These typical cases were five and a-half and three weeks old lambs, dead three and four hours respectively. Intestinal filtrates were atoxic in 5 c.c. dose in each case.

Lamb C 5.—April 30th, 1931. Four weeks old lamb died very suddenly, and examined two hours after death. The lesions were typical, and the injection of 5 c.c. intestinal filtrate killed a 3,000 gm. rabbit in five minutes.

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Lamb T 1.—May 18th, 1931. Seven weeks old lamb received twenty-four hours after death. Putrefactive changes were so marked that diagnosis was impossible. Intestinal filtrate in 5 c.c. dose caused no disturbance in a 3,000 gm. rabbit.

The filtrate from Lamb No. G 1 was available in greater quantity than that from others, and, since it had proved highly toxic to rabbits, it was used for further observation. Its toxicity to rabbits was further confirmed by the intravenous injection of 1 c.c. into each of three rabbits. In each case muscular tremors were quickly followed by convulsions and death within ten minutes in a tetanic state closely simulating that seen in affected lambs. Five c.c. of this material were injected intravenously into a normal five weeks old twin lamb During the ten minutes after injection the lamb appeared somewhat dull, thereafter it seemed uneasy, but it quickly recovered and remained normal. On intravenous injection into mice in doses of 0.1 c.c. and 0.2 c.c., this material was highly toxic.

Summary.

The filtrate from the diluted contents of the small intestine of eleven cases of pulpy kidney disease was tested as to toxicity by intravenous injection into rabbits, with the following results:— In three cases, 1 c.c. produced an early fatal result; in one case, 5 c.c. produced an early fatal result; in four cases, 1 c.c. was atoxic, and a larger dose was not tested; in two cases, 5 c.c. was atoxic, and a larger dose was not tested; in one case, 5 c.c. was atoxic in twenty minutes, but re-injection with 8 c.c. produced an early fatal result.

The injection of 5 c.c. of similar material from a lamb dead twenty-four hours previously of an undiagnosed disease, was atoxic to a rabbit.

The intravenous injection of 5 c.c. of the filtrate from one of these cases produced little disturbance in a four weeks old lamb. It was regularly toxic in doses of 0.1 c.c. and 0.2 c.c. to mice.

The Nature of the Toxic Material in Intestinal Contents.

High titre sera prepared against the toxins of *Vibrion septique*, *B. welchi* (lamb dysentery), and *B. œdematiens* were obtained from the Wellcome Physiological Laboratories; and it is a pleasure to acknowledge here the assistance of Mr. Dalling through whom this material was obtained. Simple experiments were conducted to determine whether any of these sera would neutralise the toxic properties of the diluted filtered intestinal contents obtained from Lamb No. G 1. The intestinal filtrate was mixed with an equal volume of each sera,

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incubated for half an hour at 37°C., and this mixture injected intravenously into mice. The results are set out in the Table.

Experiments with Antitoxin.

Material	Mouse No.	Dose	Result
Equal parts of *Vibrion septique* antitoxin and intestinal filtrate	7	0.4 c.c.	Dead 2 hours
	8	0.4 c.c.	Dead 4 hours
	9	0.2 c.c.	Dead 3½ hours
	10	0.2 c.c.	Dead 4 hours
Equal parts of *B. welchi* (L.D.) antitoxin and intestinal filtrate	11	0.4 c.c.	Dead 2 hours
	12	0.4 c.c.	Dead 2½ hours
	13	0.2 c.c.	Dead 7 hours
	14	0.2 c.c.	Dead 7 hours
Equal parts of *B. œdematiens* antitoxin and intestinal filtrate	15	0.4 c.c.	Dead 3½ hours
	16	0.4 c.c.	Dead 4½ hours
	17	0.2 c.c.	Dead 5 hours
	18	0.2 c.c.	Dead 4 hours
Intestinal filtrate	1	0.2 c.c.	Dead 1 hour
	2	0.2 c.c.	Dead 1 hour
	3	0.2 c.c.	Dead 1 hour
	4	0.1 c.c.	Moribund and killed 6 hours
	5	0.1 c.c.	Dead 3 hours
	6	0.1 c.c.	Dead 4½ hours

The effect of heat on the toxicity of this intestinal filtrate was also tested. After heating to 60°C. for one hour, 0.2 c.c. was injected intravenously into each of four mice and in no case did this dose give rise to any disturbance.

Summary.

Antitoxic sera prepared against the toxins of *Vibrion septique*, *B. welchi* (L.D.), and *B. œdematiens* did not neutralise the toxic property of diluted filtered intestinal contents obtained from a case of pulpy kidney disease. Exposure to 60°C. for one hour rendered this intestinal filtrate atoxic to mice.

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Discussion.

Towards the close of this work (May 9th, 1931) the writers received from Gill a private communication detailing his investigations of the 1930 (October–November) pulpy kidney season in New Zealand. Since then a short popular summary of these investigations has been published.[4] One of his lines of work was the investigation of the toxicity of the intestinal contents of lambs just dead of the disease. In a proportion of suitable cases the supernatant from the diluted centrifuged contents of the small bowel contained a substance highly poisonous to mice on intravenous injection. Three out of four lambs intravenously injected with this material died showing typical symptoms and at post-mortem examination showed lesions indistinguishable from those of naturally occurring cases.

A toxic property was not regularly demonstrated in the filtrate from diluted intestinal contents of lambs dead of this disease in North Wales, but it may well be that had larger doses been tested some of the negative filtrates would have been found toxic. It seems reasonable to assume that if a toxin of intestinal origin is responsible for these deaths, its concentration in intestinal contents will be far from constant, being high in those " acute " cases of short duration, and low in lambs in which the illness has been of some duration.

These two sets of observations can only be regarded as preliminary, and nothing more than the most tentative conclusions may be drawn from them. They do, however, suggest that a study of the toxicity of intestinal contents in a long series of cases of pulpy kidney disease would be profitable, and might determine the nature of this condition.

It is interesting, with the little evidence available, to speculate on the possible nature of the toxic substance present in the intestine of these cases. Gill apparently favours the view that it is a bacterial toxin. In his recent private communication he records the observation that one of the toxic intestinal contents was thermolabile, and not neutralised by *B. welchi* (L.D.) antitoxin. One of the toxic filtrates isolated in North Wales was thermolabile, and not neutralised by antitoxic sera prepared from *V. septique*, from *B. welchi* (L.D.), and from *B. œdematiens*. The fact that this material was rendered atoxic by exposure to 60°C. for one hour fits the theory of bacterial origin; but the neutralisation experiments suggest that, if bacterial, it is not the product of one of the commoner anaerobes. The writers are at the moment rather inclined to view it as a product of protein disintegration. It may well be that, in the high degree of metabolic activity characteristic of the class of lamb attacked, and the un-

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balanced diet of rapidly growing young grass, the digestive processes are so unhinged that a toxic product, or toxic products, of protein breakdown are released in considerable amount, sufficient to cause the death of the lamb. The observation that filtrate is easily rendered atoxic by exposure to heat does not discountenance this possibility since certain such products are unstable.

The field observation that the injection of serum prepared against *B. welchi* (L.D.) in very early life appears to protect lambs against pulpy kidney disease did at first suggest that a member of this group, living in the intestine, might be responsible for the condition. It hardly seems likely, however, that such an injection could retain much of its specific protective power for some three or more weeks. Rather would it seem that these injections could only have the effect credited to them if the foreign protein element, horse serum, were regarded as the active part.

General Summary.

(1) The condition chiefly responsible for the deaths among young forward lambs three to six weeks old in North Wales appears identical with the pulpy kidney disease described in New Zealand by Gill.

(2) The primary predisposing conditions in North Wales are the age of the lamb, its markedly forward condition, and the quick growth of spring grass. These factors are closely interrelated.

(3) Field observations make it appear that the injection of lamb dysentery serum at birth has markedly reduced loss from this pulpy kidney disease.

(4) In a proportion of the cases investigated, during the spring of 1931, diluted filtered intestinal contents were highly toxic on intravenous injection to rabbits and to mice. Such material from one case produced only mild transient symptoms on injection into a normal four weeks old lamb.

(5) Antitoxic sera prepared against the toxins of *Vibrion septique*, of *B. welchi* (lamb dysentery), and of *B. œdematiens*, did not neutralise the toxicity of intestinal filtrate, but exposure to 60° C. for one hour rendered this filtrate atoxic to mice.

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THE CAUSATION OF STONE IN INDIA.*

By ROBERT McCARRISON, C.I.E., M.D., D.Sc., Hon. L.L.D., F.R.C.P.

Honorary Physician to the King; Colonel, Indian Medical Service; Director, Nutritional Research, Indian Research Fund Association.

URINARY calculus is very prevalent in India. From reports, for which I am indebted to civil surgeons, it appears that, during the years 1926–28, 34,325 persons were found to be suffering from the disease; or 1 in 10,000 of the population. These represent the combined records for three years, and relate only to persons seeking medical relief; but when it is remembered that hospital patients form but a relatively small fraction of the people of India, and that hospitals and dispensaries are but sparsely distributed, it may safely be assumed that the disease is more prevalent than these figures indicate.

The incidence of stone varies greatly in different parts of the peninsula; it is so prevalent in some localities that they are known as " stone-areas," while in others it is rare or unknown. The prevalent belief that India in general is a land of " stone " is erroneous: parts are; others are not. Thus, taking the above figures as our index of its prevalence, we find such variations as the following:

CASES PER 100,000 OF POPULATION.

In India generally	10
In Dera Ghazi Khan (Punjab)	438
In Hyderabad (Sind)	266
In Sukkur (Sind)	156
In Manipur (Assam)	84
In Dera Ismail Khan (N.W.F.P.)	62
In Saharanpur (U.P.)	25
In Ahmednagar (Lower Bombay) ...	13
In Benares (U.P.)	5
In Sylhet (Assam)	2.8
In Malabar (Madras)	0.3
In Kolaba (Lower Bombay)	0

Of the 34,325 sufferers from stone, 16,384, or approximately 1 in 20,000 of the population, had their stones removed at operation.

* The second of two lectures on " Experimental Research " at the Pasteur Institute, Coonoor, South India, delivered before the Royal College of Surgeons of England, May 20th, 1931. Reprinted from the *British Medical Journal* by kind permission of the Editor.

Many hints of great value to veterinary clinicians, especially to those who specialise in canine practice, will be found in the text between pages 415 and 430.—ED. V. J.

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I have taken this figure as my index of incidence and prepared the spot map (Fig. 1). Each spot represents one stone removed at operation per 100,000 of the population of the particular district in which the patients lived. This map affords a fair indication of the distribution of the malady. It will be noted that the great "stone-area" of India extends from Sind, the North-West Frontier Province, and the Punjab into the western parts of the United Provinces. As we pass

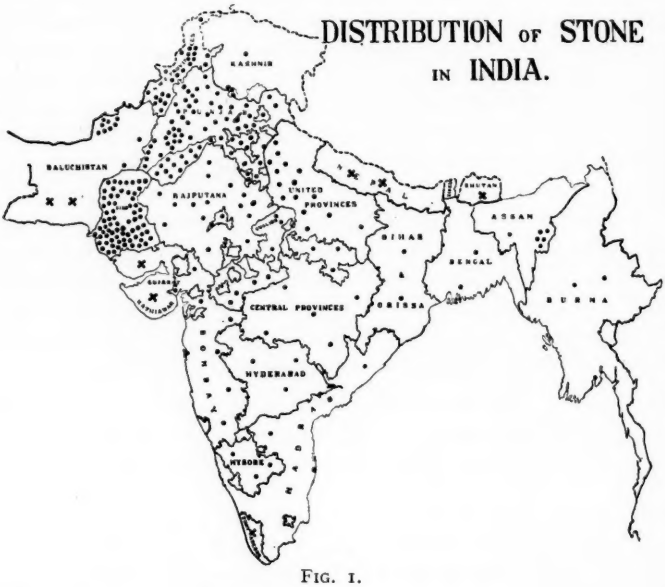

FIG. 1.

eastward towards Bengal and Assam the prevalence of the disease gradually diminishes; similarly, it diminishes towards the south and south-east until in the southern parts of the Bombay Presidency, Mysore, Hyderabad, and Madras, the malady is relatively rare. It is relatively rare also in Burma. No reports were received from the greater part of Baluchistan, which abuts on Sind, and in which there is reason to believe the disease is also common; nor from Kathiawar, nor Travancore in the south. But these lacunæ do not influence the general result, which, from the point of view of the investigator, is a significant one. It is that there are local factors which exercise a profound influence on the occurrence or non-occurrence of the malady. These influences may be geological, orographical, climatic, racial, nutritional, or microbic.

Geological Influences.

Urinary calculus occurs for the most part on soils of the Post-Tertiary period; indeed, a map of the geological features of India shows a great Post-Tertiary band extending eastwood from the Kirthar, Sulaiman, and Hindu Khush Mountains on the west and north through the cultivated plains of sub-Himalayan India to Bengal and Assam; and comprising the great plains of the Indus, the Thar or Indian Desert, regions eastward of "the five rivers" (Punjab), and the plains of the Ganges and its tributaries. It is in these plains that the great majority of all urinary calculi occur. At first sight one might be inclined to attribute significance to this association with Post-Tertiary rocks. But closer scrutiny of our data shows that no great importance can be attached to it, since it is in the plains of the Indus, and not in those of the Ganges, that the great majority of calculi occur.

Orographical Influences.

While stone is most common in the plains of Northern India, it is by no means confined to them. Far up in the mountain ranges of Dardistan, and throughout the Himalaya, one finds it also. In Chitral and Gilgit, in Gizar and Ishkoman, I have, in my early days, performed many operations for the relief of the condition—conscious even then of the temporary nature of the relief I was affording the sufferer. Stone is not related in any conspicuous way to the physical features of the country.

Climatic Influences.

It is most frequent in the hot, arid, desert plains, where vegetation is relatively scanty; where grazing for cattle is poor, and scorched by the intense heat, or parched because of scanty rainfall. But its incidence is not always greater in these localities than in others more favourably situated. Thus it occurs with great intensity in the cultivated lands, watered by the five rivers of the Punjab or by the numerous Himalayan streams that go to swell the waters of the Ganges. Nor does rainfall appear to be a factor of importance: in Dera Ismail Khan (N.W.F.P.)—that furnace of the plains—where the rainfall is rarely more than five inches throughout the year, the incidence of stone is 62 per 100,000 of the population, as compared with 84 in Manipur (Assam), where the rainfall is 100 inches, or thereabouts, and vegetation plentiful. One thing the western and eastern areas of the great stone-belt have to some extent in common: a great disparity between summer and winter, and between day and night, temperatures. But here again too much importance can be

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attached to this fact. It has been suggested that, because of the known effect of hypervitaminosis-D in causing stone in rats, the constant exposure of a large part of the body to the rays of the tropical sun may induce in natives of India a condition of hypervitaminosis-D, conducive to stone formation. Experiments on albino rats have not, in my hands, lent support to this view. Further, the exposure is greater in the loin-clad southerner than in the more completely clad northerner.

Racial Influences.

It is stated in certain monographs on " stone " that Mohammedans in India are more subject to it than Hindus ; and it has been suggested that this is due to the Mussulman's habit of micturating in the squatting position, which renders complete evacuation of the bladder impossible. I have no data regarding the frequency of this habit in the two communities, though it would seem to be practised by both ; but whether it be true or not that the Mussulman is more addicted to it than the Hindu, my data provide no evidence that stone is more common in the one community than in the other. In my series there are 1,024 cases in which particulars are given as to the community of the sufferer : of these, 757, or 73.9 per cent., were Hindus ; 235, or 22.9 per cent., were Mohammedans ; and 32, or 3.1 per cent., were in other communities.

Hindus form, approximately, 69.5 per cent. of India's 320 million people ; Mohammedans 21.3 per cent., and other communities 9.2 per cent.—percentages which, so far as the two main communities are concerned, are practically identical with the percentage distribution of stone. Mohammedans are therefore no more subject to the malady than Hindus. If anything, other communities are more afflicted by it than either. The disease appears to be comparatively rare among Europeans in India ; in my series they formed 0.44 per cent. of the whole.

Nutritional Influences.

It is in the plains of the Indus, and in the sub-Himalayan regions fed by the upper reaches of the Ganges and its tributaries, that wheat is most grown. Here, too, the cultivation of maize is considerable ; while in Sind the chief crop is millet. As we pass eastward along the Gangetic plain the cultivation of rice increases, reaching its acme in the Delta ; southward, also, through Lower Bombay to Malabar and Madras, and south-eastward to the deltas of the Godaveri and Kistna Rivers, the cultivation of rice increases. In Burma, too, rice is the principal, and often the only, cereal produced. In these

places rice is the staple article of diet. The suspicion thus arose in my mind that the gradual decline in the incidence of stone, from regions where wheat is the principal cereal grown to those where rice is the chief crop, might be associated in some way with the cereal forming the staple article of diet of the people. Experiments on animals showed that this was so. Groups of young albino rats were fed on a diet of the same composition but in which the cereal, forming 90 per cent. of the diet, differed. It was found that cereal foods ranged themselves in the following descending order of stone-producing potency: whole-wheat flour, oatmeal, North Indian millet (*bajra*), white flour, rice, and South Indian millet (*cambu*). The result of this experiment provided a partial explanation of the peculiar distribution of stone: its great frequency in the wheat-growing areas of the north-west, and its relative infrequency in the rice-growing areas of the south and east. But this association is not invariable. Thus, stone is as common, even commoner, among the rice-eating Manipuris (Assam) as in the wheat-eaters of Dera Ismail Khan (N.W.F.P.). There is more in the occurrence of stone than the cereal which happens to be the staple food in this or that part of India: other dietetic factors are concerned. What these factors are has been revealed, in some part at least, by experiment on animals.

Experimental Production of Stone.

Well-fed albino rats do not suffer, certainly not in Coonoor, from stone; and these animals are very suitable for experimental work on this disease. I have used them throughout my own work. My first experiment was begun early in 1926. It lasted 157 days. Such experiments are tedious; the more so when one's object is to ascertain the faults in Indian dietaries to which "stone" might be attributable. The diet used consisted of oatmeal 53 parts, linseed meal 20 parts, cornflour 25 parts, calcium phosphate 1 part, sodium chloride 1 part, and distilled water *ad libitum*. It may be wondered why I used oatmeal, since it does not enter to any considerable extent into the dietaries of the Indian people. I confess at once that chance determined my use of it—a lucky circumstance such as occasionally comes the way of the investigator, and for which he must ever be on the watch. The original object of the experiment was not to produce stone, but to learn the effect on the thyroid gland of an iodine-poor diet such as this one was. But as things turned out, the experiment taught me three things: (1) that the so-called iodine-poor diet did not cause thyroid enlargement; (2) that it did cause stone and its sequelæ; and (3) that it also caused a very grave form of anæmia,

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which subsequent investigation in my laboratory by Dr. Lucy Wills showed to be due to *Bartonella muris* carried by the lice with which my rats were infested.

Here I may pause to draw attention to this last and very instructive result: an anæmia which is brought about in practically cent. per cent. of rats by removal of the spleen is also brought about by a faulty food deficient in certain vitamins. The intact spleen protects against this anæmia; and the conclusion is irresistible that the faulty food, by its action on the pulp cells of this organ, reduces its protective action against *Bartonella muris*. This is a very noteworthy instance of the effect of faulty nutrition in breaking down the defences of the body against infection.

But to return to stone. The above diet caused the disease in 21 out of 120 animals, an incidence of 17.5 per cent. In my next experiment one group of young rats was fed on the same diet as the above, and another on a diet wherein the oatmeal was replaced by whole-wheat flour. This experiment also lasted for many days, and when at length it was completed it revealed a significant difference in the incidence of stone in the two groups: of the 50 rats fed on the "oatmeal diet" 4, or 8 per cent., had stone and its sequelæ; of the 50 fed on the "whole-wheat diet," 11, or 22 per cent., had stone and its sequelæ. Whole-wheat flour was thus shown to be more potent to cause calculus than oatmeal. The next problem which naturally occurred to me was whether the other ingredients of the diet were also factors of importance: these ingredients were cornflour and linseed meal. A further tedious series of experiments was undertaken from which the knowledge emerged that neither of these food materials contained any positive or toxic agent which was concerned in stone production. It further emerged that diets composed exclusively of white flour or of white bread, either with or without added yeast or marmite, were also capable of causing stone, though the stone-producing potency of white flour was markedly less than that of whole-wheat flour; vitamin B deficiency was not concerned in causing stone. Subsequent experiments excluded another factor—deficiency of suitable protein.

Cereal foods thus appeared to vary in their stone-producing potency, and the experiment, mention of which I have made already, was undertaken to determine their relative degrees of potency. This experiment fixed them in the following descending order: whole-wheat flour, millet grown in the north of India, white flour, rice, and millet grown in the south of India. The last did not cause "stone" at all within the 515 days of the experiment. The significance of

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this observation in regard to the distribution of stone in India has already been referred to. Here another possibility arises: millet grown in one part of India may be stone-producing, while that grown in another may not: soil, manure, and irrigation conditions—which I had already shown to be capable of causing variations in the nutritive quality of food grains (McCarrison, 1926–28)—may thus be concerned in the production of stone. I have not so far been able to investigate this aspect of the matter.

The next step in my investigations was to see whether, by further additions to the stone-producing diet, the malady could be prevented. One group of rats was therefore fed on a diet of known stone-producing potency; another on the same diet to which whole milk was added, the milk being given in the proportion of two-thirds of an ounce per rat per day. Stone and its sequelæ—cystitis, hydronephrosis, and pyonephrosis—were completely prevented by this measure. Clearly, therefore, milk contained "something" which counteracted the stone-producing action of the diet. Further experiments of a like kind followed, but in these the substance added was butter or cod-liver oil, the action of which was compared with that of vegetable oils in common use in India. The butter prevented stone, so also did cod-liver oil; the vegetable oils did not. This narrowed the issue down to deficiency of vitamin A bearing ingredients as a chief food fault concerned in stone production.

By this time I had become acquainted with the original observations of Osborne and Mendel (1917), associating urinary calculus with deficiency of vitamin A in rats fed on synthetic diets, and with the latter work of Fujimaki (1926), definitely demonstrating the specificity of vitamin A deficiency in this regard. With this help my work proceeded more rapidly, but along the lines previously followed—the production of stone by means of foods in common use by the people of India.

It was stated in some of the reports which I received from civil surgeons, as well as in text-books on the subject, that hard waters have something to do with the causation of stone. Accordingly, lime was added to diets deficient, among other things, in vitamin A, with the result that the incidence of calculus, of cystitis, of hydronephrosis, and of pyonephrosis was greatly increased; and it was found that calcium-phosphorus imbalance was a further, and very important, factor concerned in the causation of certain types of urinary calculi.

The types of stones produced in these experiments were four: ammonium-magnesium-phosphate, calcium carbonate, calcium hydro-

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xide, and stones composed of a mixture of calcium carbonate and calcium hydroxide.

My immediate object had thus been attained. It had been shown that certain food materials in common use by the people of India were capable of causing stone in rats when used in quantities disproportionate to certain other food materials that are antagonistic to stone formation.

Indian Dietaries and Stone.

In India stone is a poor man's disease. Throughout the peninsula the staple article of diet of the people is one cereal grain or another. We have seen that these cereals vary in their stone-producing potency, and it may be assumed that all cereals contain, in some degree of concentration, a *positive agent* or agents favourable to stone formation. This assumption is justified by the observation that the stone-producing potency of whole-wheat flour is greater than that of white flour—something favourable to stone production is in part removed from the whole wheat in the process of manufacturing white flour; this " something " can scarcely be vitamin A, while we know it is not any of the other vitamins. (Here it may be suggested that the lesser potency of white flour as a stone-producing agent may be correlated with the gradual diminution of vesical calculus in England during the last fifty years—a period coinciding with the introduction and almost universal use of white flour.) We have seen also that the distribution of stone in India can be correlated with the kind of cereals in use in various parts of India; and we have seen, further, that milk and butter, if used in sufficient quantity, afford complete protection against stone in rats. Now the great fault of all Indian dietaries among the poorer classes, who are the chief victims of stone, is that their diets do not contain anything like enough milk and milk products, and I postulate that according to the kind and quality of the cereal grain they use as the staple of their dietaries, and according to the poverty of these dietaries in milk and the products of milk, so they will be more or less likely to suffer from stone. Milk and the products of milk are among the greatest of India's needs at the present time. I do not contend that this is the whole story; indeed, I know it is not, but it represents certain chapters of it.

Dietetic Factors Causing Stone.

There appear, therefore, to be two categories of dietetic factors concerted in stone formation: (*a*) *positive factors* including excess of lime in the diet, and some unknown agent present in whole cereal grains; and (*b*) *negative factors*, including deficiency of vitamin A

derived from animal sources, and deficiency of phosphates relative to the amount of lime in the diet. I say " vitamin A derived from animal sources," because millions of people in India are dependent for their supply of this factor—whatever that factor implies—on vegetables; I am not yet certain that the latter source of vitamin A is, from the point of view of stone prevention, as good as the former. I do not think it is, and I notice that de Langen (1929) of the Dutch Indies is of a like opinion.

Here it is necessary to make my position clear in regard to the value of whole-wheat flour and oatmeal as articles of diet. Because I have found that whole-wheat flour and oatmeal, when they compose the major part of an imbalanced diet, are stone-producing, this is not to say that I desire to displace these two most valuable foods from the high place in the dietaries of mankind to which they have attained throughout the centuries. To do so would be to doubt the evidence of one's own eyes, and to fly in the face of reason. Who that has seen the magnificent physique of the Sikh or Pathan would dare to recommend them to change their staple diet of unleavened whole-wheat bread for the white loaf of commerce? And who that has seen the slum children at Miss Margaret McMillan's " Nursery School " at Deptford can doubt that she was right to exercise the care she did in the selection of the oatmeal on which experience had taught her to rely as a constituent of her children's dietaries—children who rarely need the dentist? The truth is that no single food material is in itself a " complete food "—even milk is not; if it be not faulty in one regard it is faulty in another. The best are those whose faults are least; and in the latter category both whole-wheat flour and oatmeal are to be included. Milk, as an exclusive food, will cause anæmia in rats; onions, anæmia in dogs; cabbage, goitre in rabbits; and oatmeal or atta will cause stone in rats; yet what better food can there be for mankind than a judicious mixture of all four? Animal experimentation has its uses—its great uses—but like every other instrument employed by man the results of its use must be judged by reason and experience.

Stone: A Disease of Faulty Nutrition.

During the course of my experimental work I have produced stone in 250 animals. The young rats that I have used have always been about a month old at the outset of the experiments; that is to say, of an age at which they are able to fend for themselves, and are no longer dependent on maternal nutriment. They would correspond approximately to children of a year or two old. I have grouped

these cases together in the chart which I now show you (Fig. 2). You will observe that in rats "stone" is a disease of early life; that the pinnacle of age incidence is reached when they are about 120 days old, when they have been living on the stone-producing diets for about 90 days. Thereafter the curve of age incidence declines

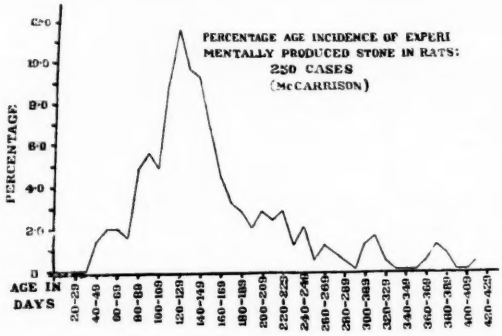

FIG. 2.

rapidly and gradually fades away until, at the age of 400 days or over, stone is relatively rare. Now compare this curve with that of the age distribution of stone in India (Fig. 3). The same rapid rise will be noticed during its earlier stages, the same rapid fall and its gradual fading away thereafter; the curves in the rat and in the Indian are practically identical. Taking these facts into consideration

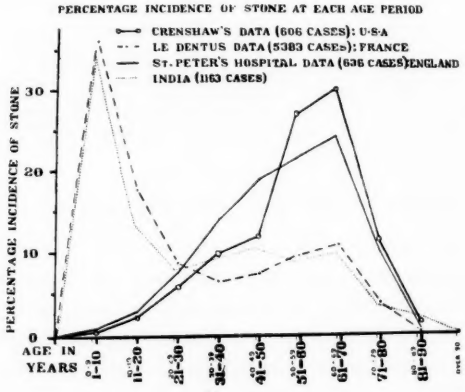

FIG. 3.

with those I have already presented, the conclusions seem to me to be irresistible that in India urinary calculus is a disease of faulty nutrition, and that the same food faults which produce the malady in rats produce it also in the people of India.

But it is not always so. In my next illustration (Fig. 3) I have set out in the same diagram the percentage incidence of stone at each age period as it is found in India, in France, in the United States of America, and in England. Here the incidence curves are of orders so different as forcibly to suggest that while the chief causes of stone in India and in France are probably the same—nutritional in the main —those in England and the United States of America are in the main different : in the one the high peak of incidence is at the earlier period of life, when nutritional disturbances are most likely to be felt ; in the other it is at the other end of life. Many factors may account for stone production in later life : stone formed in early life may be endured to later life ; stagnation of urine due to enlarged prostate and the like may cause it, or infection. But it is not my purpose to discuss the causation of stone in later life—a condition relatively rare in India.

Sex Incidence of Stone.

In India stone is much commoner in males than in females : in my series the proportion is as 13.2 to 1. In children under the age of fifteen years, 92.5 per cent. of stones occurred in males. My

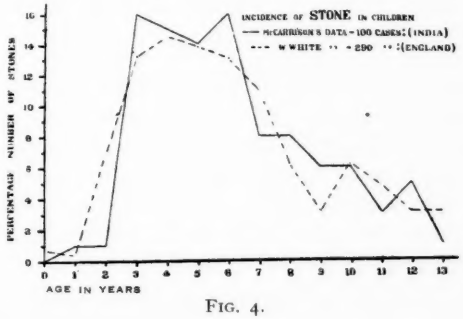
INCIDENCE OF STONE IN CHILDREN
— McCARRISON'S DATA - 100 CASES: (INDIA)
--- W WHITE " - 290 " :(ENGLAND)
PERCENTAGE NUMBER OF STONES
AGE IN YEARS

FIG. 4.

observations in this regard are in conformity with those of Winsbury White (1929)—in fact his curve and my own are practically identical (Fig. 4) ; and if I be correct in my contention that in Indian children stone is a disease of faulty nutrition, it is very likely to be so also in English children. One point of interest is brought out in this diagram

(Fig. 4) : the Indian curve shows that very occasionally stone may occur in breast-fed infants.

The greater incidence of stone in males does not apply to experimentally produced stone in rats. In these animals it is slightly more common in females. I have, at present, no explanation to offer of this difference in the two species.

Incidence of Renal Calculus in India.

In comparison with vesical calculus renal calculus is relatively rare in India (Fig. 5). In a series of 454 cases of stone recently sent to me by surgeons practising in parts of India where the disease is common, there were only 34 renal stones ; a proportion of vesical

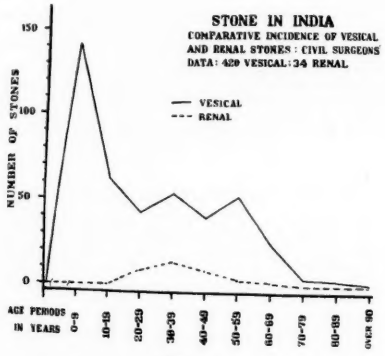

FIG. 5.

to renal of 13.5 to 1. Whereas vesical calculus is pre-eminently a disease of early life, renal caculus is a disease of middle life (Fig. 5). In the course of my experimental work I have had frequent examples of renal calculus in rats. In my series of 250 experimentally produced stones there were 31 renal calculi ; a proportion of vesical to renal of 8 to 1. In albino rats renal stone is equally common in the two sexes. In human beings it is much commoner in males.

Incidence of Ureteral Stone in India.

Ureteral stone is rare in India, or it is rarely operated upon. Among the 16,384 calculi removed during the years 1926–28 there were only 14 ureteral stones. A diagnosis of stone in the ureter was made in other cases, but the patients refused operation.

In my series of 250 experimentally produced stone in rats there were 21 examples of ureteral calculus : in the majority of these the

stone was obviously on its way from the kidney to the bladder, and was not impacted in the ureter. Males had such ureteral stones more often than females (16 to 5).

Cystitis, Incrusted Cystitis, Pyonephrosis, etc.

It is not possible, in the time at my disposal, to do more than mention the morbid states of the urinary tract so often associated with stone in experimental animals: cystitis, incrusted cystitis, hydronephrosis, and pyonephrosis. Any of these may occur without stone formation, though such occurrence is the exception and not the rule. We do not, as yet, know why one animal develops stone while another fed on the same diet develops incrusted cystitis or pyonephrosis without stone. Cystitis is not a necessary precursor of stone, even when the stone is of the ammonium-magnesium-phosphate variety. And cystitis without stone can be produced in rats with considerable certainty by diets deficient in vitamin A, but containing an excess of animal protein—a combination of food faults not uncommon in human sufferers from cystitis. Curiously enough the cystitis so produced is commoner in female than in male rats: I believe I am right in saying that in human beings cystitis is also commoner in the female sex.

Chemical Composition of Human Stones.

During the past three years analyses of 226 human urinary calculi have been made in my laboratory (Newcomb and Ranganathan). The stones may be classified according to their chemical composition as follows:

	Number	Percentage of total number of stones
Pure uric acid	15	6.63
Pure oxalate	13	5.75
Pure phosphate	3	1.32
Phosphate-oxalate	23	10.17
Urate-phosphate	20	8.85
Urate-oxalate	78	34.51
Urate-oxalate-phosphate	74	32.74
Total	226	99.97

According to Kahn (quoted by Kauffmann—1929), pure uric acid calculi are rare: they formed 6.6 per cent. of the total in our series of 226. Freyer (1908) found that most of the stones containing

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uric acid or urates occurred in young people. Our data do not confirm this observation. He also found that 64 per cent. of his Indian stones contained either uric acid or urates; our figure is higher: 82 per cent. He noted also that calcium oxalate calculi were commoner in adults than in children: in our series the average age of sufferers from pure oxalate stones was 29 years. Pfister has called attention to the predominance of oxalate stones in Egypt and in India; in our series of 226 there were 13 pure oxalate stones (5.75 per cent.), while 83.17 per cent. contained more or less oxalate.

Our analyses of kidney stones are relatively few, but they show that in India these stones consist mostly of calcium oxalate and not of uric acid or urates: an observation in conformity with that of Benjamin Moore (1911) and of Rosenbloom (1915); and in opposition to that of Stengel and Fox (1927). It would seem from the literature on this subject that renal calculi are generally considered to be composed of uric acid: this is not our experience in India.

Much difference of opinion exists as to the chemical composition of the nuclei of stones. It is not always easy to find the nucleus of a stone, or, when one has found it, to be sure that one is dealing only with the nucleus. One of my assistants (Mr. Ranganathan, 1930) has recently gone into the matter in some detail. He found that uric acid or urates formed the chief constituents of the nuclei, phosphates occurring only in traces; and that the composition of the nuclei did not always run parallel with that of the stones containing them, though an approximate parallelism existed in some cases.

There is a general belief that it is possible, from the physical characters of a stone, to make a good guess at its chemical composition. We do not find that this is so; such guesses are in general very inaccurate (Newcomb and Ranganathan, 1930).

So far as our analyses of human stones go it would seem that no particular kind of stone is more prevalent in one part of India than another. We have not found it possible, from the incomplete dietetic histories of sufferers, to correlate the chemical composition of calculi with that of the diets eaten by the people from whom they were removed: the great majority of their dietaries had, however, one fault in common—insufficiency of vitamin A bearing ingredients.

Chemical Composition of Rat Stones.

As previously stated, the stones produced in rats were of four varieties: ammonium-magnesium-phosphate, calcium carbonate, calcium hydroxide, and a mixture of the last two. The essential difference between them and human stones is the complete absence

of uric acid or urates. Excluding traces of urates (forming less than 5 per cent. of the total ingredients) uric acid or urates occur in 82 per cent. of all human stones in India. The usual explanation given for the absence of uric acid in rat stones (as well as in those of cattle) is that these animals possess the power to oxidise approximately 80 per cent. of the circulating purines to allantoin. Allantoin is several times more soluble in water than is uric acid, and hence it never forms a constituent either of rat or of cattle stones. Human beings do not possess this power, and in them the degradation of purine bodies stops short at uric acid ; and, as uric acid is very sparingly soluble in water, it is almost always found to be a constituent of urinary calculi in man (Swift Joly).

Power of the Urine to Retain Uric Acid in Solution.

Urine varies greatly in its power to dissolve uric acid and retain it in solution. We have recently found that the urine of rats fed on purely vegetable diets retains much more uric acid in solution than that of animals fed on diets containing large amounts of animal protein. Hindhede has, I believe, made a similar observation in man. The urine of rats fed on a diet of liver was capable of dissolving only a very little uric acid, and this was thrown out of solution after standing for twenty-four hours. The composition of the diet, by influencing the capacity of urine to hold uric acid in solution, may thus have a bearing on stone formation.

Chemical Composition of Cattle Stones.

Cattle stones, as we find them in India, are usually small, round, shot-like bodies, having, when fresh, a light or deep golden-yellow sheen. They may occur in large numbers (Fig. 6) in the same animal, and their usual location is the urethra. They are made up of thin layers, like the coats of an onion, all layers being of the same composition. Stones occurring in the kidney in cattle do not possess these physical characters : they are usually single, larger, of irregular shape, and are devoid of metallic sheen. But whether occurring in the kidney or in other parts of the urinary tract, their chemical composition is, with rare exceptions, the same. Analyses have been made of twenty-four cattle stones in my laboratory (Ranganathan, 1931). Their composition has been found to be fairly uniform : calcium carbonate with a little magnesium, the latter presumably also existing as carbonate. In only one of the twenty-four stones was this composition departed from. In this case (a calf) the stone had a relatively high moisture content, a high nitrogen, phosphate, and magnesium content, and a low calcium content ; carbonates were not present.

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We have been fortunate in being able to reproduce not only the chemical but the physical (excepting the metallic sheen) characters of cattle stones under experimental conditions in albino rats (Figs. 7, 8). Briefly, this is how it was done. The rats were fed on a diet deficient in vitamin A, and containing an excess of lime relative to the amount of phosphate in the diet (McCarrison, 1931). By keeping them on this diet for seventy to eighty days, and then supplying the missing vitamin A (in the form of radiostoleum) it was found that of

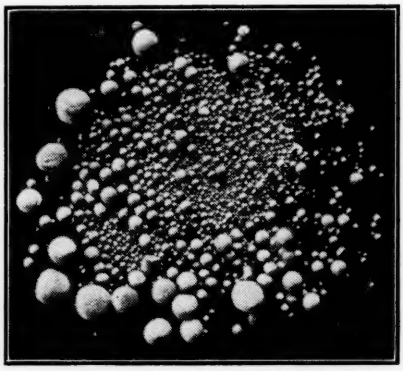

FIG. 6.—Urinary calculi in cattle.

those that survived the process a high percentage had stones of composition and physical characters closely resembling those of cattle stones. Apparently what happens is this: During the first, or vitamin-starvation, period an enormous mass (Fig. 7) of calcium carbonate debris is deposited in the urinary tract. On supplying vitamin A, but allowing the calcium-phosphate imbalance to remain, the whole (if it be small) or part (if it be large) of the calcium carbonate deposited during the first period is redissolved and dispersed. But in some cases parts remain as isolated collections or grains; and during the second period of the experiment, when vitamin A is provided, these act as foreign bodies, and a layer or layers of calcium carbonate is deposited around them. This is at present a working hypothesis; but we hope by hitting off just the right diet to keep the rats alive long enough, and by alternating the periods of vitamin-want and of vitamin-supply with sufficient nicety, while retaining the calcium-phosphate imbalance, to reproduce these cattle stones with still greater exactitude.

In chemical composition the stones experimentally produced in rats were identical with the calcium carbonate stones found in Indian cattle (McCarrison, 1931).

Through the kindness of Mr. Hilson, Director of Agriculture, Madras, I have been able to secure reliable information regarding the food of the cattle in parts of the Madras Presidency where cattle stone is prevalent. They are fed on maize, millet, or rice straw,

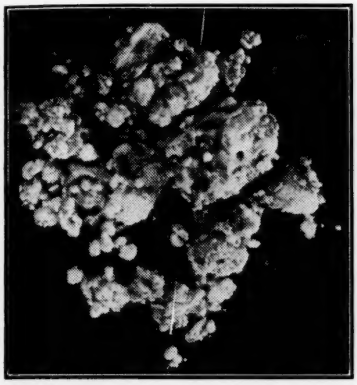

FIG. 7.—Calcium carbonate concretions weighing 1,530 mg. removed from the kidneys of rat.

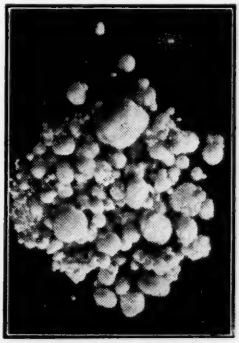

FIG. 8.—Calcium carbonate stones weighing 715 grams removed from the bladder of rat fed on a diet deficient in vitamin A and phosphate and containing an excess of lime. Compare the latter with the stones removed from the urinary tract of a bullock (Fig. 6).

the husks of ground nuts, and the empty pods of legumes. All these fodders are poor in phosphates and disproportionately rich in lime; ground-nut husks, for instance, contain as much as 3.78 per cent. of CaO and only 0.36 per cent. of P_2O_5. After the rains, the cattle obtain much more green fodder, and with it a much-needed supply of vitamin A. Periods of vitamin-want alternate with periods of vitamin-supply, the diet remaining all the while imbalanced with respect to calcium and phosphates. It is notable that the only exception to the rule that cattle stones are composed of calcium carbonate was in a calf, aged six months, which, after weaning, was fed on a diet in which calcium phosphate imbalance was not marked. Strong support is thus lent to the hypothesis above mentioned as to the manner of calcium carbonate stone formation. Our investigations

have shown, further, that in the formation of these stones the calcium phosphate imbalance is even more important than the vitamin A deficiency (Ranganathan, 1931). We find that the incidence of such stones can be very greatly reduced in rats by adding to the vitamin-A-poor and lime-rich diet an amount of sodium phosphate sufficient to form, with the excess of lime, insoluble calcium phosphate in the bowel. The further addition of vitamin A completely prevents the disease. I may therefore claim with some reason that the problem of " cattle stone " in India has been solved so far as the laboratory can solve it.

Function of Vitamin A in the Prevention of Stone.

In my last lecture I referred at some length to the effects of vitamin A deficient diets on epithelial structures throughout the body. No tissue suffers more severely from want of this vitamin than that of the urinary tract. It seems to me very probable that desquamated, keratinised epithelium from this tract may form the nidus round which calculus deposition occurs. It is true that in rats infection, as a rule, rapidly supervenes ; and this, no doubt, may be in part responsible for the chemical composition of one variety of rat stone—the ammonium-magnesium-phosphate variety. But although naked-eye evidence of infection is the rule, exceptions occur in which this variety of stone is found without any obvious cystitis. Further, cystitis is no less common in rats suffering from calcium carbonate stones, and in these ammonium-magnesium-phosphate is conspicuous by its absence.

In my experiments I have found that the volume of urine excreted depends to a great extent on the presence or absence of vitamin A bearing ingredients in the diet ; when absent or small in amount the volume of urine is low ; when present in sufficient quantity it is normal or high. Sodium phosphate has also a very marked effect in this regard, greatly increasing the urinary excretion. It is significant that associated with the addition of vitamin A bearing substances (whole milk, cod-liver oil, or radiostoleum) to stone-producing diets, and of these substances *and* sodium phosphate to such diets containing an excess of lime, the urinary excretion from being scanty becomes copious, and at the same time stone is prevented.

There is, of course, another direction in which vitamin A acts in the prevention, and its deficiency in the causation, of stone. I refer to its power to prevent infection of the urinary tract, and to the influence of its deficiency in favouring such infection. This brings me to the last of the influences which may determine the occurrence and distribution of stone in India—infection.

Infectious Infections.

The work of Rosenow and his colleagues of the Mayo Foundation is no doubt familiar; his experimental production of gall-stones in animals by infection with streptococci from cases of cholecystitis in man; and, more recently, his production of urinary calculi by the devitalisation and infection of teeth in dogs with streptococci from cases of nephrolithiasis. Rosenow's procedure is, first, to produce "septic foci," using, for the disease he wants to produce, streptococci obtained from a case of the particular disease in man. He finds the coccus to be specific; that from cholecystitis will produce gall-stones; that from nephrolithiasis, urinary calculus. This sequence of events is probably the same in essentials as that occurring in rats: in these, foci of devitalisation are produced in the urinary tract by the defective diet, and, in some forms of stone at least, chance infection may do the rest. But the point of special interest to India in Rosenow's work is that a specific kind of infection may, as he and Meisser suggest, be associated with stone, and thus account for the prevalence of the disease in certain localities. Personally, I am quite prepared to believe that this may, to a limited extent, be so; for although I have shown that the distribution of stone in India may, in some part, be accounted for by the kind of cereal used by people whose diets contain an insufficiency of vitamin A bearing ingredients, this is not the whole story, nor does it explain the occurrence of stone in all localities in India, or in all individuals. Bilharzia, though a frequent cause of stone in Egypt, does not appear to be so in India; and in infection by bilharzia malnutrition is a potent favouring influence.

Mechanism of Stone Production.

This lecture has dealt with the causation of stone, not with the mechanism of calculus deposition. My researches have been mainly of the practical kind: my business being to tell the people of India how they can themselves avoid stone and prevent it in their cattle. It matters little to them, though it is of great interest to the medical scientist, whether the deposition of stone can be most satisfactorily accounted for by faulty metabolism due to dietetic causes, by infection, or by physico-chemical disturbances in the urinary tract. I have little doubt that both faulty food and infection may induce the physico-chemical disturbances responsible for stone deposition; but I know that a properly constituted diet will go a long way to prevent both infection and physico-chemical disturbances. It seems to me that all three agents—dietetic, infectious, and physico-chemical—play their part in the genesis of stone, and in designing its architecture;

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on occasion, and in certain individuals or in certain places, the star part is taken by one or by the other. In India the star part is taken by diet.

The President and Council of the College have signally honoured me by their invitation to tell something of my research activities in India, and I have been deeply conscious of the responsibility this honour has imposed upon me. I have purposely chosen to refer only to those of my activities which I have thought might be of special interest to surgeons. Even with this limitation much has had to be omitted or passed by with but a few words. But if what I have said may serve to indicate directions in which the newer knowledge of nutrition touches upon the Art of Surgery—to indicate lines of investigation which hold out the prospect of the solution of certain surgical problems—then I shall have fulfilled, in some small measure, the duty imposed upon me by the invitation to address this great College.

PIG ANÆMIA.

By L. P. DOYLE.

Purdue University, Agricultural Experiment Station, LaFayette, Indiana.

ANÆMIA in suckling pigs is coming to be recognised as an important disease. In the past it unquestionably caused important pig losses without the true case being recognised. Early experiments at the Indiana Agricultural Experiment Station showed that keeping sows and their pigs inside of central hog houses or barns continuously until the pigs were two to five or six weeks old produced severe anæmia in the pigs, while allowing the animals to have access to outside conditions gave protection against the disease. The earlier experiments with anæmia also showed that there was a very striking relationship between severe anæmia and a high death loss in suckling pigs. In some experiments, the death loss was as high as 65 per cent. in anæmic pigs, while it was as low as 3 per cent. in the non-anæmic pigs.

More recent experiments have shed considerable light on the specific factors in outside conditions which prevent anæmia. Sunlight did not appear to be of much direct importance. Access to soil, however, particularly soil bearing blue grass sod, proved to be highly effective in preventing the disease. Where blue grass sod was fed to sows and their pigs, the young animals were solidly protected against anæmia although they were kept inside continuously.

Pigs which were separated from their mothers for three to five hours daily and placed on blue grass sod inside of a central hog house escaped anæmia ; while their litter mates which did not have access to sod, developed a severe form of the disease. Pigs which were placed on soil free from sod did not fare as well as those placed on sod, but they fared much better than the ones which did not have access to either sod or soil. The feeding of green feed (green rye, green alafalfa and green oats) to sows and pigs which were kept inside in concrete pens failed to show any well-defined preventive effect on anæmia in the pigs. In this experiment the feeding of green feed (soiling) was begun at the beginning of the period of gestation, and was continued until the pigs were four weeks of age.

The beneficial effect of allowing the pigs to range over blue grass pasture while the sows were kept confined in small outside pens, was strikingly shown by an experiment in which twenty-two litters were used. All of the pigs were farrowed inside of a central hog house. Eleven of the sows and their litters were kept inside continuously until the pigs were four weeks old. The other eleven sows and their pigs were put outside when the pigs were three to six days of age. The sows which were put outside were kept confined in small pens with cinder floors, and provided with small shelter houses, but the pigs were allowed to range over a small blue grass pasture. The amount of hæmoglobin in samples of blood from each pig was determined at weekly intervals, beginning when the pigs were born and continuing until they were four weeks old. The pigs which were kept inside showed a continuous decrease of hæmoglobin, while the ones which had access to blue grass pasture showed a slight decrease at the end of the first week after birth, and then a continuous increase until the normal level was reached at four weeks of age. The pigs which had access to pasture had an average of 13.0, while the pigs which were kept inside had only 5.5 grams of hæmoglobin per 100 c.c. of blood when four weeks of age.

What may prove to be of great practical significance in connection with the prevention of anæmia is the observation that the disease failed to occur in two litters of pigs farrowed by sows which had run on blue grass pasture during the gestation period, and which had been feed sparingly on skim milk and yellow corn. These were put in a central hog house a few days before farrowing, and they and their pigs were kept inside continuously until the pigs were eight weeks old. The two litters comprised fifteen pigs—seven in one litter and eight in the other. The fifteen pigs made normal growth during the eight weeks of inside confinements, and none of them became anæmic.

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The ration which was fed to the sows and pigs during the period of confinement was the same as had been used repeatedly in producing anæmia under inside conditions.

The results of preliminary experiments in which blue grass sod was fed to sows kept inside during pregnancy suggest that the feeding of sod, with limited amounts of a suitable ration, may have a preventive effect on anæmia in the pigs, even when the use of the sod is discontinued at farrowing time.

Clinical Articles

A Case For Diagnosis.

By H. LLEWELYN JONES, M.R.C.V.S.
Denbigh.

On January 9th this year I received an urgent telephonic message to go and see a mare which had aborted the previous night, and that she was unable to move, and sweating profusely. On my arrival at the farm, which was thirteen miles away, I was given the following history: Owing to the very hard frost which was, and had been, in existence for some time, the horses had not been out at work. On the previous day, however, the mare in question, a light vanner, ten years old, which was due to foal the end of May, was taken out in a light cart to a mill about one and a-half miles away. Travelling was very difficult and dangerous, and made all the more so because the mare was of a very nervous temperament, and always frightfully energetic, with the result that she became very frightened, and perspired freely, but she managed the journey to all appearances and she was normal that evening, but on the following morning they discovered that she had aborted and parted with the afterbirth, and was standing in the stall bathed in perspiration, breathing heavily and unable to move either foreward, backwards, or sideways. That was the picture which presented itself to me as I entered the stable. Temperature was 105.6° F.; pulse, 72—quick, hard, and wiry; visible mucous membranes were of a dirty-red colour; practically all the muscles of the body quivering, and there were frequently repeated jerky contractions of the muscles of the hind and fore limbs, but more especially the hind ones. The actual standing position was not far from normal, but the two hind feet were too far apart, and the muscular

spasms were very sudden. A sample of the urine was taken and sent the same day to Professor Hare, Royal Veterinary College, London.

It was turbid, brown in colour, and contained numbers of reddish-brown floccules in suspension. It had no distinct odour, was amphoteric to litmus, and had a specific gravity of 1025. It was positive for albumen and for blood ; but negative for sugar, kenones, bile salts, and bile pigment. The deposit on centrifugalisation was moderate in amount and brown in colour, and on microscopic examination consisted of large numbers of red blood corpuscles, amorphous material, triple phosphate crystals, and a few epithelial cells. Uterine exploration revealed nothing abnormal under the circumstances, and on emptying the rectum the fæces were also normal.

Diagnosis.—When I received the phone message I thought it might be a laminitis, but when I saw the mare I could see at once it was not. After my examination, the next thing was to " give it a name." If it was an ordinary case of azoturia, how was it the mare was not attacked before she reached home, or even later in the evening ? And how was it that the mare was still standing when I first saw her ? In the ordinary course of events one would find the patient down and unable to rise, with the muscles of the hind quarters hard and tense—but they were not. Though the symptoms somewhat resembled those of azoturia, I could not bring myself to believe that it was, and I told the owner that it was a case of tetany, but after events proved that I was wrong.

Treatment.—As I was of the opinion that it was a case of tetany, to be logical, I had to apply " the " treatment. Fortunately I had the milk-fever outfit with me, so I inflated the udder, and in addition inflated her subcutaneously at three different places on each side until she was beautifully blown up all over. Unfortunately, I had no calcium gluconate with me, and I could not return for it there and then.

I administered an ounce of liq. formaldehyde, and four ounces of sodium chloride in a gallon of water with the stomach pump, and just before I left I gave her a six-dram physic ball, and some diuretic powders, to be given in the form of an electuary. It was impossible to move the mare, so I had to leave her in the stall, with a couple of warm rugs on. On the following morning I was much surprised to find her no better, and standing in exactly the same spot. I still had hopes the calcium glucose would bring about a speedy recovery, and she received two ounces of the crystals dissolved in a quart of boiling water (and for this purpose I keep the ordinary aluminium hot-water bottle and a Higginson syringe, and never use them for anything else except the various hypodermic and

intravenous injections). It was injected as usual at six different sites: three on each side, behind the scapula, over the scapula, and on the ends of the neck. A great deal of sterilising is needed each time (bottle, syringe, etc.), but I think it pays, as so far I have been fortunate enough never to experience sloughing. The solution is injected as near body temperature as possible, and I never inject the drugs from the bottle, for there is often a little sediment there by the time one has finished and the solution has cooled down. They were told to groom her twice daily, and keep her well clothed, with plenty of barley water to drink. She refused all kinds of foods.

Prognosis.—Inflation having failed, or, in other words, seeing that I had made a mistake in diagnosis, I told the owner that the case, if it recovered at all, would take weeks, and it did—months!

On the 12th I saw her again, and as she had gone down during the night, it was with the greatest of difficulty that they got her up, and into a loose box. There was a slight improvement in temperature, pulse, and colour of the membranes and urine. For several days after this she either stood in the same spot, constantly picking up one hind foot, then the other, jerkily, or lying in a flat, recumbent position. For a long time she had to be assisted into the natural lying-down position before she could get up, which she did without further help. The appetite by now had improved, but she developed huge bed sores. She was given iodide of potassium and carbonate of iron in her feed, and liquor arsenicalis in the drinking water. As time went on she gradually improved, but the sudden flexion of the hind legs, especially the left one, continued for five weeks. The discoloration of the urine did not disappear for nearly three weeks. Towards the middle of February she was placed on gentle walking exercise for a few minutes twice daily, and the string-halt action of the hind limbs would to a great extent disappear after walking twenty yards or so. All attention now was concentrated on grooming, dieting, and exercising. By March 10th the bed sores had healed, and the mare looked much better, but a trace of the peculiar jerky action of the near hind off limb was still to be noticed. During the months of May and June the mare was out at grass, and she has now made a complete recovery, having put on an enormous amount of flesh, and doing light farm work.

If any of your readers feel inclined to express their views on the case through the medium of the journal or directly with me, I should be delighted. I am still sceptical about azoturia, for there were many features in the case which certainly do not make their appearance in an ordinary attack of that disease.

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Milk Fever Three Months After Calving.

By J. V. S. RUTTER, M.R.C.V.S.
Hereford.

Subject.—" Maiden," a red, shorthorn cow, in milk-recorded herd. Ten years old, had eight calves, never been ill previously. Calved January 3rd, 1931, and since then had averaged six gallons of milk daily. In her 1930 lactation period she gave 1,117 gallons of milk, and during 1929, 1,339 gallons. Her food ration was regulated by her milk yield, *i.e.*, she received 3½ lb. " concentrates " (consisting of equal parts by weight of oats, linseed cake, and barley meal) for every gallon of milk. Her average daily ration would therefore contain about 21½ lb. concentrates, plus 17 lb. hay, and fresh water *ad lib.* With other cows she was turned out to graze for a short period each day whilst the cow sheds were being cleansed and disinfected.

History.—6.30 a.m., April 1st, 1931 (N.B.—three months after calving). Had not cleared up concentrates, but had eaten all her hay. She was turned out with the other cows, and appeared a little wild, which made the cowman think that she was coming " in season." Her milk yield had decreased 25 per cent. 1.30 p.m.: Cow down in stall, but in normal posture. Disinclined for food, but apparently chewing her cud. Appeared rather " full," and would not rise to her feet, so the attendant gave her a dose of mag. sulph. c̄ zingiberis, xii oz. Soon afterwards I was telephoned for, being informed that the cow was badly " blown," and could not get up.

3.30 p.m.—I examined the cow and found her temperature normal, respirations accelerated and shallow; pulse slightly quickened, and reflexes normal. She was certainly tympanitic, but not excessively so. She appeared to be chewing her cud, but on close observation I was able to see that this was really only a nervous movement of the jaws, with some slight grinding of the teeth. Grunting occurred when the abdomen was " kneaded." The probang was passed, and a little gas released; this was followed by ol. tereb. c̄ aether meth., given per probang, and this brought away more gas. The cow was turned on to her other side, and a further quantity of gas was released, but she still refused to rise. I then gave ¾ gr. strychnine sulphate hypodermically, and applied stimulating liniment to the loins. She was rugged up, and small doses of liq. ammon. fort. c̄ aether meth. left to be given her at two-hour intervals until she recovered. I instructed the cowman to let me know if she got any worse, otherwise I would visit her in the morning.

11 p.m.—Informed by phone that the cow was worse and in much pain. 11.30 p.m.: On entering the byre I heard the characteristic milk-fever "moan," the cow was lying with her nose tucked into her flank, and she was grunting and moaning with each breath. The corneal reflex was impaired, there was excessive salivation and champing of the jaws, and she was unable to rise. Her temperature was 102 degrees, and her pulse and respirations quickened. The tympanites had disappeared. I diagnosed milk fever, and as such inflated the udder, and gave adrenalin hypodermically; more liniment was applied to her loins. We packed the cow up comfortably in a sitting position with straw, etc. On leaving I told the cowman on no account to drench her, but to apply the liniment to the loins every two hours until she was on her feet.

April 2nd, 3.30 a.m.—The cow rose to her feet of her own account and looked for food, afterwards making an uninterrupted recovery. For the following week she was kept on a restricted diet, but during the next week she was gradually brought on to normal food, and at the same time she gradually returned to her normal milk yield. Since that time she has been in perfect health.

Stomach Staggers.

By FRANK CHAMBERS. F.R.C.V.S.
Wolverhampton.

THE expression "stomach staggers" is a term used to describe cases of cerebral disturbances in cattle which may or may not end fatally. It is a bad term, for the condition has little to do with the state of the actual stomach. However, the term will be used until a better one has been coined to describe more accurately or scientifically this condition.

During the month of May this year I had an outbreak on a large sewage farm. The outbreak first started on May 14th, when the farm manager found a cow dead in the field. He had her carted away by the horse slaughterer, but no post-mortem was made. When the cows were brought in on the next morning one was observed to be very ill. She was delirious, and eventually got down. She died within half an hour of being seen. I was then sent for. Examination of the blood for anthrax being negative, I made a post-mortem, but could find nothing wrong. Later on in the day two more cows were found to be affected, and the manager inflated their udders. When I saw the one cow, a large roan, she was sitting up, but could

not get to her feet ; she had apparently got over the very acute symptoms. The other cow a red, six-year-old, was on her side, frothing at the mouth and executing galloping movements with her legs ; she was semi-unconscious. I re-inflated the udder, and injected intravenously 150 c.c. of a 10 per cent. solution of calcium gluconate. This cow got up the same afternoon and went on all right. On May 16th, I had the roan cow killed as she made no effort to get up. On May 17th, another cow became affected. She was given an udder inflation by the manager. I saw her later, and as she was very shaky on the legs, gave her a subcutaneous infection of calcium. This cow recovered. On May 17th, a light roan cow was found down in the field, on her broadside, and semi-conscious. Her udder was injected, and an intravenous injection of calcium gluconate given. She recovered consciousness in an hour's time, but would make little or no attempt to get up. Several injections of calcium were given, but with no effect. This cow was eventually slaughtered on May 22nd, and a careful post-mortem made. On May 18th, two more cows showed slight symptoms, but udder inflation, plus a mild purgative, put them both right. The symptoms shown in all cases resembled milk fever very closely. The two animals that lingered on were never really alert, and would, if allowed, fall on to their sides.

At the time of the outbreak the cows were grazing on sewage land that had been flushed two weeks previously with effluent rich in ammoniacal liquor, but no effluent had been run on since that date. The grass was very luxuriant, and the cows on being turned into this field ate ravenously. They were moved off immediately after the second case occurred, and were put on a pasture that never had sewage on, and which was comparatively bare. It was thought that the pasture on the first meadow would be very rich in nitrogen, and this had upset the calcium balance in the cows' blood. Although the cows were taken off the rich pasture, sporadic cases occurred, the last on June 26th, when a cow that had calved four weeks previously was found down in the field and semi-conscious. She was inflated by the manager, and got to her feet, only to go down again four hours afterwards. I was then asked to see the animal, and found what appeared to be a typical case of milk fever, and I injected 60 c.c. of dextro-calcium. She got to her feet eight hours after the injection, but was found down again on the 28th. This time I tightly inflated the udder, and gave a subcutaneous injection of adrenalin. She got to her feet two hours afterwards and went on well.

During the height of the epidemic I called in Mr. R. Trigger, and together we made a post-mortem on one of the cows that had been

down two days. As is usual in these cases, there was nothing to be seen which could be called pathological. A sample of the blood was sent to Professor Russell Greig, who reported that the analysis showed a calcium concentration of 5.5 mmg. per cent. This is about a 50 per cent. reduction from the normal, and represents a marked hypocalcæmia. Dr. Greig, in his his letter to me, stated : " It seems that you are dealing with an acute intoxication, but it is not obvious that the calcium deficiency is a primary factor in the disease. It may be secondary to the intoxication, but this is mere speculation." It was observed that in most of the cases, especially the mild ones, there was a marked diarrhœa. Since mammary inflation, combined with calcium injection, only brought about a response in a small proportion of the cases, the condition was not identical with milk fever. At this time I had a number of similar cases on other farms. The analysis of the stomach contents by Dr. Clough was absolutely negative, and a further analysis of the sewage effluent failed to reveal the presence of any mineral poison. I am of the opinion that this outbreak was due either to the absorption of fodder over-rich in protein, in which case it is possible that intermediate or toxic products of protein decomposition were absorbed ; or the upsetting of the calcium-phosphorous balance, due to the effluent which is known to be rich in phosphates, these being excreted *via* the urine, thus forming an important component of sewage.

Vegetable proteins contain amino-acids which, when metabolised, liberate toxic bodies like benzoin acid, phenol, indol, etc., and these are normally excreted either as hippuric acid or ethereal sulphates. If the mechanism governing this protective function is disturbed a toxæmia occurs ; an excess of protein may be sufficient alone to cause this disorganisation, and consequently it is not impossible that this was what occurred in the above cases. Unfortunately, I was not able to carry out urine analysis, but interesting data might have been obtained.

Ringworm Communicated from Cattle to Man.

By H. BUCKINGHAM, M.R.C.V.S.
Norwich.

A NUMBER of years ago I attended fifteen half-grown bullocks, every one was suffering more or less from ringworm. I gave each animal a ball and a draught, and dressed the lesions. About ten days afterwards my right arm began to itch, and upon looking at it

I found five circular spots of ringworm. I showed it to my doctor, who gave me some ointment to apply. This did no good; in fact each lesion increased in area, some being the size of a two-shilling piece, and others as big as a five shilling piece. Some coalesced.

It caused me great pain when I was administering balls to horses, cattle and sheep. I became impatient, and told my doctor so, and he then applied chrysophanic acid. This was painful, to say the least of it, but it cured the disease.

Stockings Swallowed by Cattle.

By H. BUCKINGHAM, M.R.C.V.S.
Norwich.

I WAS once called to see five cows who had broken into the drying ground where the good lady had put about twelve pairs of woollen stockings on the line to dry. The cows had swallowed them all. I administered copious doses of linseed oil, turpentine, and Epsom salts. Not one of them was seriously ill. One passed a large piece of a stocking per rectum.

Aricyl in Small Animal Practice.

By H. E. SMITHERS, M.R.C.V.S.
Edgware.

ARICYL can, with advantage, be used in a great variety of diseased conditions in the dog. In fact it is not too much to say that there are very few conditions that are not benefited by its administration. Aricyl is an organic solution of arsenic with a complex formula. It has all the properties of arsenic, that is to say, it is a hæmatinic, nervine tonic, stimulant to the gastric mucosa (improving appetite and digestion), and general alterative. The action of aricyl on the gastric mucosa may be doubted, but in the writer's opinion it is a fact. Before discussing individual cases, of which a dozen have been selected, a mention of the mode of administration found to be most effective might be of interest. The subcutaneous route has been found quite satisfactory, and no abscess formation has ever been seen. Five doses of 1 c.c. are given for five consecutive days, and, if necessary, another five are given at intervals of three days until ten doses have been administered.

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Early in 1928, the writer first began to use aricyl, giving an occasional dose to dogs that were not feeding, and so on, but it was not until May, 1928, that the drug was used as described above. This was when *Case* 1 came for treatment. The cases have been grouped according to the disease rather than in chronological order.

Case 1.—Liver spaniel bitch, aged six months, suffering from partial paralysis, accompanied by circus movement. Aricyl was administered, 1 c.c., as already described, and the animal was discharged cured. She has since borne two litters of pups.

Case 2.—Alsatian bitch, aged eighteen months, when first seen was a typical picture of distemper. Three weeks after the distemper symptoms had abated she had one fit and became partially paralysed in her hind quarters, and later developed chorea in the left fore leg. Aricyl cured the paralysis, but signs of chorea can still be seen, especially when the bitch comes in œstrum.

Case 3.—Liver and white springer spaniel, aged nine months. Developed complete paralysis two weeks after apparent recovery from distemper. He was quite recovered after ten injections of aricyl.

Case 4.—Sheep dog, aged one year. This case was not seen until after it had recovered from the usual clinical symptoms of distemper. It was reputed to be having seven or eight fits a day, and was partially paralysed. Pot. bromide was given to control the convulsions, and aricyl injections were commenced at once. After two days it was reported that the fits had ceased, so pot. bromide was discontinued. The aricyl was administered as usual, and at the end of the course the animal was completely recovered.

Case 5.—Black Alsatian bitch, aged eight weeks. Suffering from general anæmia and debility, and complete anorexia. Aricyl was administered, and after three days the mucous membrane of the mouth and eyes which, when first seen, were paper white, appeared pinker, and after five doses the colour of the mucous membrane was normal, and the animal had recovered its appetite. It has since grown to be quite a respectable specimen of its breed.

Case 6.—Wire-haired terrier dog, aged three months. Listless, not feeding, emaciated, and generally showing signs of debility. After ten doses of aricyl he was playing, feeding, and behaving like a normal puppy.

Case 7.—Wire-haired terrier dog, aged about two years. Suffering from a form of gastritis. He vomited everything, including medicine. So aricyl was given a trial. He was quite recovered after five doses.

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Case 8.—Mongrel dog, about six months. A case of typical clinical distemper. This was the first distemper case for which aricyl was adopted with no other treatment. The active symptoms subsided after five doses had been given, and there were no sequelæ of any kind.

Case 9.—Wire-haired terrier dog, aged nine months. Another distemper case. This case needed the full ten doses of aricyl, but there were no untoward sequelæ.

Case 10.—Alsatian bitch, aged eighteen months, suffering from distemper. Treated with aricyl, and made a complete recovery.

Case 11.—Mongrel dog, aged two years, suffering from dry eczema. This case resisted all treatments until aricyl was tried. All scabs, etc., disappeared a week after the last dose of aricyl.

Case 12.—Mongrel dog, aged six years. Skin irritation and erythema. Irritation and erythema disappeared after aricyl, but reappeared again this spring.

Acute Endocarditis in the Dog.

By E. J. HEATHER, M.R.C.V.S., D.V.S.M.
Oxford.

THESE three cases of acute endocarditis of the benign type in the dog are selected from a certain number as being typical in my experience of the course of the disease from its onset. This experience has caused me to make slight differentiations from the common lines of treatment, and the usually accepted course of certain symptomatic disturbances.

The subject of *case A* was a cross-bred whippet-collie dog, twelve years old, which had been in robust health the whole of its life except for a slight gastric disturbance some two years previously. Without any apparent cause it developed slight malaise, became lethargic, lost appetite, and within thirty-six hours was evidently a very sick dog in dire need of skilled assistance. Upon clinical examination an enormously accelerated pulse-rate, somewhere in the region of 210, and a tumultuous action of the heart which was such a medley of sound that it was impossible to ascertain any definite rhythmic disturbance, were the most obvious symptoms of trouble. The temperature was 104°, and a most striking feature of the general uneasiness of the animal was its persistent refusal to assume a recumbent position.

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Mild febrifuge and stimulant treatment was adopted, and during the three days that followed the acuteness of the symptoms subsided. Coincident with this remission, respiration was slightly accelerated, until, seventy-two hours after the first clinical examination was made, it was approximately one and a-half times the normal rate of the dog. At this time the temperature had returned to normal, pulse-rate was 140, and it was possible to distinguish more definite sounds upon auscultation of the heart. The first sound of the heart was obscured by a systolic murmur, and an increase in the force of the beat had occurred. The animal was by this time in a more comfortable state. Appetite and spirits had improved, and it was beginning to resume much of its old routine General improvement steadily continued, and fourteen days afterwards examination found the pulse to be at the normal rate of 100, rather small in quality but quite regular, while auscultation revealed the systolic murmur, which had been observed before covering the first sound, to be more distinct. The great force of cardiac contraction was evidenced by the easily observed movements of the heart within the thoracic cavity. A typical picture of aortic stenosis was thus furnished. Treatment was reduced to the regular administration of digitalis, alternating with strophanthus in conjunction with aromatic stimulants. Twenty days after onset of the disease, anæmia and emaciation of a progressive nature commenced. This continued for the next nine weeks until the animal, although in good though somewhat subdued spirits, and in no obvious discomfort, was in an extremely poor bodily condition. Attacks of vertigo every few hours ensued, and the animal was destroyed at this stage, as it was obvious that natural decease was only a matter of a few days.

Case B presents features of rather a different nature. The subject was a Labrador retriever dog, nine years old, which had a perfectly clean record as regards previous illness, and was in the best of health at the onset of disease. Without warning, one day the animal developed malaise, loss of appetite, fatigue after slight exercise, and frequent panting attacks, and a subdued cough at intervals. Upon examination it was found that a temperature of 105° persisted, respiration was approximately one and a-quarter times normal rate, though this varied considerably upon gentle exertion ; the pulse was rapid, varying between 160 and 180, and auscultation of the heart revealed a forcible cardiac contraction, with a systolic murmur entirely obscuring the first and almost all of the second sound of the heart.

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This state of affairs continued for seven days, increasing somewhat in intensity of symptoms in that period. After the seventh day there was a decline of temperature, respiration rate became slightly slower and more regular, cough increased, pulse rate fell to 140, and the systolic murmur was confined more to the first sound of the heart, the second sound becoming louder and more forcible. At this stage anæmia and wasting, accompanied by abdominal dropsy, made their appearance. Attacks of vertigo and total refusal of food occurred at intervals of every third day. These were usually followed by brighter periods, in which spirits returned to a marked degree. A typical picture of pronounced mitral insufficiency was presented.

Digitalis, alternating with strophanthus in combination with mild diuretics, was prescribed with satisfactory results. The general symptoms were alleviated and the animal existed in a quite comfortable state for several weeks. After twelve weeks, however, progressive wasting and anæmia had reduced the dog to a very poor condition, and at the same time the amount of abdominal œdema had gradually increased in spite of temporary remissions. Dyspnœic attacks became more frequent owing to diaphragmatic pressure of the fluid, and it was during one of these attacks that *paracentesis abdominis* was resorted to.

One hundred ounces of fluid were removed gradually, but no relief was given, and the amount was replaced with interest within three days. The animal was destroyed at that juncture, sixteen weeks after onset of the disease.

Case C presents features in common with both the preceding cases. The subject was a black, long-coated retriever bitch, seven years old, with no history of any previous illness. Onset of disease was sudden and acute, the most marked symptoms being a rise of temperature to 104 degrees, acceleration of respiration, increase of pulse to 160, immediate appearance of abdominal dropsy, and a total refusal of the animal to lie down for four days. Symptoms of anæmia and wasting which appeared almost at the onset became less progressive as time went on, and heart sounds became diagnostic of a pronounced degree of aortic stenosis, with a lesser amount of mitral insufficiency.

At the time of writing, ten weeks after the first symptoms became manifest, the animal is in a very similar condition to what it was eight weeks ago. There is, if anything, a slightly greater degree of mitral insufficiency with, as far as can be ascertained by clinical examination, the same degree of aortic stenosis. Further wasting of musculature, and increase of abdominal œdema has been arrested, and the animal

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appears to have the prospect of an indefinite span of life in its present condition.

These three instances are typical of what I have encountered in cases of this disease in dogs, and, among other points, would lead one to the conclusion that, firstly, the disease does not depend upon a previous, predominating bloodstream infection for its birth, but that the ætiology is of a more specific infective nature ; secondly, that the acuteness of the primary infection determines the degree of valvular damage from the onset, not that the lesions are produced through the heart in a mechanically progressive manner, starting from the results of a pathological state of one group of valves after the acute stage of the disease has subsided.

Enterotomy for Faecal Impaction.

By CLIFFORD FORMSTON, M.R.C.V.S.

Department of Surgery, Royal Veterinary College, London.

THE subject, a fox-terrier bitch, eight years old, was brought to the College on June 18th, 1931, with the following history : It had been ill for three days, off its feed, and vomiting at irregular intervals. The vomit consisted of mucous and fæcal matter. There was no inclination to drink water or any other liquid. She had not passed fæces for five days although numerous doses of purgative medicine had been given. The bitch was in good bodily condition : temperature 103° F., but the breath was very foul smelling.

On abdominal palpation a chain of hard irregular lumps could be felt. Digital examination revealed an empty rectum. The owner was told that the condition might either be one of alimentary obstruction, or a chronic closed metritis, although there had been no vaginal discharge. Permission was given to perform an exploratory laparotomy.

The bitch was admitted to the infirmary, and a soap-and-water enema given ; but apart from prolonged straining nothing happened. Under morphia, gr. $\frac{1}{4}$, and general anæsthesia, an exploratory operation was performed. The uterus was found to be more or less normal. The small colon, however, was impacted for a distance of twelve inches. From the anus to the hindermost part of the impaction (a distance of ten inches) the bowel was empty. As manipulation failed to move the mass posterior of the impacted mass, enterotomy was decided upon. The incision was made over the last two inches of

the area of bowel concerned. It was then found that there were adhesions between the fæcal matter and the bowel mucous membrane. These were broken down and the fæces extracted. The rest of the bowel contents were removed by manipulation through the incision.

The bowel was closed with Czerney Lembert sutures of fine catgut. After-treatment consisted of liquid foods for several days. Healing of the abdominal wound was by first intention, and at the end of eight days the bitch was discharged from the infirmary fit and well. The owner brought the bitch to the College ten days ago, and it was then in excellent health.

Examination of the removed bowel contents revealed numerous small pieces of bone, and the owner informed me that the bitch had been accustoned to eat chicken bones without ill-effect previously.

Lipoma in the Pelvis.

By J. J. DAVIES, M.R.C.V.S.
West Ham.

An Airedale bitch, nine years old, was brought to the surgery with a history that for the previous two or three days she had been listless, refusing food, and continually straining, but unable to pass anything. On examination, two globular swellings in close contact to each other were visible to the left of the base of tail. The tumours were soft, and fluctuated somewhat, whether palpated from the outside or from the rectum. Abscess formation or a prolapsed bladder was suspected, but on thrusting a hypodermic needle into both no fluid could be obtained. Examination of the abdomen followed. This revealed the bladder as a tense oval tumour the size of a cocoanut.

The bitch was then hobbled on to a table, and the bladder catheterised. Blood-coloured urine flowed through the catheter, at times it merely trickled through. It took twenty minutes to obtain four and a-half pints. About half-way through this operation the patient started breathing heavily, developed slight tremors, and was evidently in great distress. These symptoms gradually became more pronounced, the pulse became weak and "running down." The bitch was destroyed.

Post-mortem and Conclusions.—The tumours at the base of the tail proved to be two lipomata, each being about 1½ in. in diameter, and connected by an isthmus of identical tissue, 3 in. long, ¼ in. thick. They were in contact with the rectum laterally, vagina inferiorly,

and peritoneum anteriorly. The bladder was ruptured, the abdominal cavity being full of bloody urine. The alarming symptoms during catheterisation were undoubtedly a sequel to rupture of the bladder, which probably occurred when the animal was fastened on the table in the abdominal position.

An Unusual Gestation.

By W. BEDDARD, M.R.C.V.S.

On August 10th I was called to a sow which the owner stated had commenced to farrow the evening before, and when I arrived I found a fully developed but dead piglet in the vaginal passage. This I removed in pieces, as it was tightly fixed, and the sow gave no help.

The interesting part, however, is that she had farrowed quite naturally exactly four weeks before, and had eight well-grown month-old piglets sucking on her in the usual way. The one I removed on the 10th was fully formed, and gave me the impression that it had died at the normal time of farrowing. The owner stated that the sow had never been sick nor sorry.

Abstracts of Current Literature

Blanc and Caminopetros.—Transmission of Fowl Pox by Flies.—*Rev. Gén. de Méd. Vét.*, August.

After several investigations the authors came to the following conclusions :—

(1) Flies of the species *Culex pipiens*, having bitten cockerels affected with avian variola, can transmit the malady by the bite to healthy cockerels, and this at least fifty-eight days after being infected, that is to say, probably during the whole duration of life.

(2) *Culex pipiens*, having bitten pigeons affected with pox, can transmit the malady by their bite to healthy pigeons at least thirty-eight days after being infected, therefore probably all their life.

(3) Avian variola transmitted by the bite of *Culex*, both in the cockerel and pigeon, takes the same clinical form as in nature.

(4) The bite of an infected *Culex* can be followed in the cockerel and pigeon by infection not discernible clinically, but sufficient to give immunity.

Scomorokhoff.—Vaccination against Foot-and-Mouth by the intra-cutaneous method.—*Soc. de Biologie*, January 4th, 1930.

THE author tried to realise immunity against aphthous fever by means of intradermal vaccination. He used the formalised vaccine recommended by Vallée, Carré and Kinjard. The inoculation was made in the skin of the neck, or by scarification of the labial mucosa. One of the three tests made deserves attention because it was carried out in ordinary conditions of practice, and on twenty-four bovines. Five days after one intradermal injection of formalised vaccine of 2.5 c.c. all the animals were placed in contact with an affected herd; none of them contracted the disease. This experience seems to indicate that this procedure may be of service in the fight against foot-and-mouth disease.

Michailoff.—The Treatment of Warts in the Dog with Yeast.—*Tierarztliche Rundschau*, 41.

YEAST contains a series of ferments (maltose, anylose, glucose, and simose), a vitamin B, and, further, a proteolytic ferment that kills bacteria and has an antitoxic effect. In veterinary medicine it is used in distemper, strangles, pneumonia, white scour, gangrenous skin affections, and eczema of the horse and dog, and acne. The author has used it with success on twelve out of thirteen dogs affected with warts in the mouth and pharynx. Fluid yeast was given to the animals three times daily in tablespoonful doses (small animals a dessertspoonful). The warts disappeared in three to eight weeks. No digestive disturbance was noticed as result of the yeast.

Gerlach, F.—New Researches on the Tubercle Bacillus B.C.G. *Rev. Gén. de Méd. Vét.*, July 15th, 1929.

INOCULATIONS made by Gerlach on more than a thousand animals of different species, by various routes, show that in massive doses the stock of B.C.G. causes certain tuberculous modifications in test animals; these lesions remain localised, reach their highest point of development some weeks after inoculation, and then decline and heal completely.

Successive passages of the same strain on the guinea-pig do not increase its virulence. On the contrary, this attenuates quite quickly in the cultures.

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It is also known, according to Petroff, that it is possible to separate in the cultures of B.C.G. two types of colonies, one of which is harmless and the other, named colony S, sometimes developing on the edges of the culture medium, proves virulent for guinea-pigs and rabbits. The tests of Gerlach show, on the contrary, that colonies S of an original Petroff culture do not differ in any way as regards their degree of virulence from the entire culture of B.C.G.

Finally, the author has practised vaccination following the Calmette-Guérin method on 375 new-born calves in a tuberculous milieu, and many have already been re-vaccinated. A certain number have been slaughtered, and have not shown any trace of tuberculosis at the autopsy. Those which live actually develop exceptionally well, have a very good appetite, are very lively, and nothing allows them to be distinguished from non-vaccinated healthy calves. These observations attract particular interest from the fact that in the region where they have been made it was not possible to rear calves on account of the enormous losses from tuberculosis.

News

As a result of the "Eclipse" Ball, organised by Lady Chesham in aid of the Royal Veterinary College Re-building Fund, the sum of £2,649 19*s*. 7*d*. has been handed over to the treasurer.

Publishers' Notices

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